# FOREIGN ANIMAL DISEASES

## USDA Select Agent and Toxin List

<table>
<thead>
<tr>
<th><strong>USDA only agents and toxins</strong></th>
<th><strong>USDA/HHS overlap agents and toxins</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Livestock</strong></td>
<td>• Bacillus anthracis</td>
</tr>
<tr>
<td>• African horse sickness virus</td>
<td>• Botulinum neurotoxins</td>
</tr>
<tr>
<td>• African swine fever virus</td>
<td>• Botulinum neurotoxin producing species of Clostridium</td>
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<tr>
<td>• Akabane virus</td>
<td>• Brucella abortus</td>
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<tr>
<td>• Avian influenza virus (highly pathogenic)</td>
<td>• Brucella melitensis</td>
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<tr>
<td>• Bluetongue virus (exotic)</td>
<td>• Brucella suis</td>
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<tr>
<td>• Bovine spongiform encephalopathy agent</td>
<td>• Burkholderia mallei</td>
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<tr>
<td>• Camel pox virus</td>
<td>• Burkholderia pseudomallei</td>
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<tr>
<td>• Classical swine fever virus</td>
<td>• Clostridium perfringens epsilon toxin</td>
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<tr>
<td>• <em>Cowdria ruminantium</em> (Heartwater)</td>
<td>• Coccidioides immitis</td>
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<tr>
<td>• Foot-and-mouth disease virus</td>
<td>• Coxiella burnetii</td>
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<tr>
<td>• Goat pox virus</td>
<td>• Eastern equine encephalitis virus</td>
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<tr>
<td>• Japanese encephalitis virus</td>
<td>• Francisella tularensis</td>
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<tr>
<td>• Lumpy skin disease virus</td>
<td>• Hendra virus</td>
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<tr>
<td>• Malignant catarrhal fever virus (exotic)</td>
<td>• Nipah virus</td>
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<td>• Menangle virus</td>
<td>• Rift Valley fever virus</td>
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<tr>
<td>• <em>Mycoplasma capricolum</em> /M. F38/M. mycoides capri (contagious caprine o pleuropneumonia)</td>
<td>• Shigatoxin</td>
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<tr>
<td>• <em>Mycoplasma mycoides mycoides</em> (contagious bovine pleuropneumonia)</td>
<td>• Staphylococcal enterotoxins</td>
</tr>
<tr>
<td>• Newcastle disease virus (VVND)</td>
<td>• T-2 toxin</td>
</tr>
<tr>
<td>• Peste des petits ruminants virus</td>
<td>• Venezuelan equine encephalitis virus</td>
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<tr>
<td>• Rinderpest virus</td>
<td>• Shigatoxin</td>
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<tr>
<td>• Sheep pox virus</td>
<td>• Staphylococcal enterotoxins</td>
</tr>
<tr>
<td>• Swine vesicular disease virus</td>
<td>• T-2 toxin</td>
</tr>
<tr>
<td>• Vesicular stomatitis virus (exotic)</td>
<td>• Venezuelan equine encephalitis virus</td>
</tr>
</tbody>
</table>
OIE – World Organisation for Animal Health

List A

Transmissible diseases that have the potential for very serious and rapid spread, irrespective of national borders, that are of serious socio-economic or public health consequence and that are of major importance in the international trade of animals and animal products. *(OIE does not use the List A/List B classification any more)*

- Foot and mouth disease
- Swine vesicular disease
- Peste des petits ruminants
- Lumpy skin disease
- Bluetongue
- African horse sickness
- Classical swine fever
- Newcastle disease
- Vesicular stomatitis
- Rinderpest
- Contagious bovine pleuropneumonia
- Rift Valley fever
- Sheep pox and goat pox
- African swine fever
- Highly pathogenic avian influenza

List B

Transmissible diseases that are considered to be of socio-economic and/or public health importance within countries and that are significant in the international trade of animals and animal products.

**Multiple species diseases**

- Anthrax
- Aujeszky’s disease
- Echinococcosis/hydatidosis
- Heartwater
- Leptospirosis
- New world screwworm *(Cochliomyia hominivorax)*
- Old world screwworm *(Chrysomya bezziana)*
- Paratuberculosis
- Q fever
- Rabies
- Trichinellosis

**Cattle diseases**

- Bovine anaplasmosis
- Bovine babesiosis
- Bovine brucellosis
- Bovine cysticercosis
- Bovine genital campylobacteriosis
- Bovine spongiform encephalopathy
- Bovine tuberculosis
- Dermatophilosis
- Enzootic bovine leukosis
- Haemorrhagic septicaemia
- Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis
- Malignant catarrhal fever
- Theileriosis
• Trichomonosis  
• Trypanosomosis (tsetse-borne)

Sheep and goat diseases

• Caprine and ovine brucellosis (excluding B. ovis)
• Caprine arthritis/encephalitis
• Contagious agalactia
• Contagious caprine pleuropneumonia
• Enzootic abortion of ewes (ovine chlamydiosis)
• Maedi-visna
• Nairobi sheep disease
• Ovine epididymitis (Brucella ovis)
• Ovine pulmonary adenomatosis
• Salmonellosis (S. abortusovis)
• Scrapie

Equine diseases

• Contagious equine metritis
• Dourine
• Epizootic lymphangitis
• Equine encephalomyelitis (Eastern and Western)
• Equine infectious anaemia
• Equine influenza
• Equine piroplasmosis
• Equine rhinopneumonitis
• Equine viral arteritis
• Glanders
• Horse mange
• Horse pox
• Japanese encephalitis
• Surra (Trypanosoma evansi)
• Venezuelan equine encephalomyelitis

Swine diseases

• Atrophic rhinitis of swine
• Enterovirus encephalomyelitis
• Porcine brucellosis
• Porcine cysticercosis
• Porcine reproductive and respiratory syndrome
• Transmissible gastroenteritis

Avian diseases

• Avian chlamydiosis
• Avian infectious bronchitis
• Avian infectious laryngotracheitis
• Avian mycoplasmosis (M. gallisepticum)
• Avian tuberculosis
• Duck virus enteritis
• Duck virus hepatitis
• Fowl cholera
• Fowl pox
• Fowl typhoid
• Infectious bursal disease (Gumboro disease)
• Marek’s disease
• Pullorum disease

Lagomorph diseases

Bee diseases
• Myxomatosis
• Rabbit haemorrhagic disease
• Tularemia

• Acariosis of bees
• American foulbrood
• European foulbrood
• Nosemosis of bees
• Varroosis

**Fish diseases**

• Epizootic haematopoietic necrosis
• Infectious haematopoietic necrosis
• Oncorhynchus masou virus disease
• Spring viraemia of carp
• Viral haemorrhagic septicaemia

**Mollusc diseases**

• Bonamiosis
• Haplosporidiosis (H. nelsoni or H. costale)
• Marteiliosis
• Mikrocytosis (Mikrocytos mackini)
• Perkinsosis

**Crustacean diseases**

• Taura syndrome
• White spot disease
• Yellowhead disease

**Other List B diseases**

• Leishmaniosis

### Differential Diagnosis of Vesicular Diseases

<table>
<thead>
<tr>
<th></th>
<th>Cattle</th>
<th>Sheep</th>
<th>Swine</th>
<th>Horse</th>
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<tbody>
<tr>
<td>FMD</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>R</td>
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<tr>
<td>Vesicular stomatitis</td>
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<tr>
<td>Vesicular Exanthema of Swine</td>
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<tr>
<td>Swine Vesicular Disease</td>
<td>R</td>
<td>R</td>
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</tbody>
</table>

Horses are susceptible to Vesicular Stomatitis but resistant to the other vesicular diseases
SELECTED FOREIGN ANIMAL DISEASES

FOOT AND MOUTH DISEASE

IDENTIFICATION
1. Foot and Mouth Disease (FMD) is a contagious, viral infection primarily of cattle, swine, sheep, and goats, but also of other cloven footed animals. It is characterized by vesicular lesions, and subsequently erosions, of the epithelium of the mouth, nares, muzzle, feet, teats, udder and rumen pillars.
2. Foot and Mouth Disease is caused by a picornavirus.
3. Last outbreak of FMD in the United States was 1929, Canada 1952 and Mexico 1954. FMD is endemic in much of S. America, Europe, Africa and Asia. Japan, Australia and New Zealand have been free of the disease for at least 50 years.

SIGNS
Clinical Features.
1. The clinical signs and lesions of FMD, VS, VES, and SVD resemble each other so closely that a field diagnosis is almost impossible to make. The classical signs of all these vesicular diseases are salivation and lameness caused by the formation of vesicles or blisters in the mouth and on the feet. Dullness, inappetence, a fall in milk yield, and fever usually precede the appearance of vesicles. Later quivering, drooling, smacking of lips, nasal discharge and kicking or lameness are evident. These signs may become worse after the vesicles rupture.
2. Morbidity very high in susceptible populations. Rarely fatal in adult animals (<5% mortality) but responsible for marked production loss. Mortality due to myocarditis may be high in young animals which sometimes die without showing premonitory signs.
3. Diagnostic lesions are epithelial vesicles or blisters which rupture to leave large eroded areas. Lesions are found in the mouth, lips, muzzle or snout, coronary band and interdigital spaces, teats, udder, rumen pillars and in the myocardium and skeletal muscles.

With the exception of rumen, myocardial and other muscle lesions, Vesicular Stomatitis, Vesicular Exanthema of Swine and Swine Vesicular Disease produce lesions in the same sites as FMD.
4. Incubation period usually 3 - 5 days in natural infections.
5. Uncomplicated cases recover in about 2 weeks, but sequelae may include secondary infection of lesions, chronic mastitis, chronic unthriftiness. Anestrus and other breeding problems may last for months.
**Post Mortem Examination**
Apart from the vesicles mentioned, extensive myocardial lesions of degeneration and necrosis may be seen in young animals. In severe cases these may be in stripes or bands (“Tiger Heart”).

**Differential Diagnosis**
The typical vesicles with blanched epithelial covering, filled with clear, colorless or straw colored fluid are pathognomic of FMD, VS, VES, or SVD. As the lesions age they may resemble lesions appearing in other diseases such as IBR, Malignant Catarrhal Fever, Rinderpest, BVD/Mucosal Disease, photosensitization, bovine herpesvirus mammilitis etc.

**Field Samples**
Contact State and Federal Veterinarians.
1. Vesicular Fluid
2. Epithelial covering or flaps from vesicles

Freeze samples except for blood.

**Laboratory Diagnosis**
Detection of viral antigen by ELISA test. Confirmation can be obtained within 3 hours. Typing by genome analysis to determine a suitable vaccine within 3 days.
There are 7 recognized serotypes of the disease of which some are geographically restricted.

**Control**
Low risk countries including the United States have a “stamping out” policy. High risk countries or those where the disease is endemic have vaccination programs using inactivated mono-, bi- or tri-valent vaccines.

**SWINE VESICULAR DISEASE**

**IDENTIFICATION**
Swine Vesicular Disease (SVD) is a contagious viral infection of swine. It causes vesicular lesions and, subsequently, erosions of the epithelium of the mouth, nares, snout and feet.
Also caused by a **picornavirus**. Related to the human enterovirus Coxsackie B-5. The disease was first recognized in Italy in 1966 and for the next ten years caused confusion in various European countries because of its clinical resemblance to FMD. In 1973 a meeting of the European Commission for the Control of FMD established standard diagnostic procedures. The disease has occurred outside Europe in Taiwan and Japan.
SVD has economic importance in its own right because affected hogs become lame, lose appetite and weight and become unthrifty. It also causes interference with international trade, since many countries ban import of pigs or pork products from countries where the disease occurs.
SIGNS
The clinical signs and lesions of Swine Vesicular Disease are indistinguishable from FMD.
Subclinical disease may occur, resulting in failures to detect the disease and complicating eradication procedures.
Swine are the only species affected. (But note in using this as a diagnostic clue: some strains of FMD do not readily infect cattle).

Post Mortem Examination
Vesicles as in FMD with evidence of damage to the feet being seen up to 30 days after recovery. Extensive microscopic lesions including perivascular cuffing and infiltration by PMN leukocytes.

Field Samples
Same as FMD

Epidemiology
Endemicity of SVD will severely hinder early detection of FMD.
SVDV is a very stable virus and is not inactivated by acid changes in musculature after death. Dried salami and pepperoni prepared from the meat of infected swine can contain residual virus for at least 200 days.

VESICULAR STOMATITIS

IDENTIFICATION
Vesicular stomatitis is a disease of horses, cattle and swine manifested by vesicles in the mucosa of the gums, tongue, muzzle and skin of the coronary band and interdigital area. The agent is a bullet shaped rhabdovirus of the genus vesiculovirus. There are two pathogenic serotypes, New Jersey and Indiana.
Vesicular Stomatitis is a Western Hemisphere disease occurring in North and South America and the Caribbean. It is the primary differential diagnosis for FMD in the USA.

SIGNS
In swine and cattle the disease is clinically indistinguishable from FMD. In milking cattle the teats are often the most severely affected site. Mortality is low, and uncomplicated recovery is usual

Diagnosis
Differential diagnosis includes bluetongue, which affects cattle, although it is more severe in sheep and does not affect horses. On premises where a vesicular disease affects cattle and horses VS is the most likely diagnosis but beware: FMD and VS have been found simultaneously in the same herd.
Collect the same field samples as for FMD and SVD

Epidemiology & Transmission
Wild life reservoirs appear to exist in the tropics and sub-tropics with the virus cycling in forest mammals. Sandflies (Simuliidae) have been implicated in transmission and transovarial infection occurs. Explosive outbreaks occur north and south of the endemic areas and are difficult to control.
Transmission can occur between pigs but seems to be unusual between cattle.

**Control**
The primary method of control in the USA is to **quarantine** affected premises until 30 days after the disappearance of clinical signs.

**The last major outbreak in the USA was in May 2010 in Arizona. This is a disease you may well see.**

### VESICULAR EXANTHEMA OF SWINE

**DEFINITION**
This is an acute febrile disease of swine with vesicles appearing around the snout, lips and coronary bands. Primary lesions may appear within 24 hours of exposure with secondary lesions occurring in 2-4 days.

**VES** and a number of other clinically indistinguishable diseases of swine are caused by **Caliciviruses**.

VES spread from California to all the major swine producing areas of the USA between 1939 and 1952. In 1954 a federal eradication program was started and the disease was last seen in the USA in 1956. It is now considered a foreign animal disease.

Many caliciviruses of marine animals are known and a calicivirus isolated from calves on the Oregon coast is capable of causing vesicular lesions in pigs.

**SIGNS**
Vesicular Exanthema mimics FMD, VS, and SVD. Abortion, agalactia, runting, myocarditis and encephalitis have also been reported. Morbidity is variable but mortality is low. The major impact is in unthriftiness which makes pork production unprofitable.

**Post mortem examination**
Vesicles as in FMD and occasional myocarditis and encephalitis.

**Diagnosis**
All vesicular diseases affecting swine are on the differential list. Differentiate by laboratory diagnostic tests. See FMD for samples to collect.

**Epidemiology**
Swine are probably an aberrant host for what are primarily a group of marine viruses. In the last major epidemic transmission was through feeding uncooked swill. Certain fish are now recognized to carry caliciviruses infectious for swine and thus cooking of swill, fish or e.g. seal meat before feeding to swine is required.

Some of these viruses are infectious to primates and should be regarded as having zoonotic potential.

**African Horse Sickness**
Midge borne Orbivirus genus of the Reoviridae. Nine known serotypes. Characterized by fever and edema of the subcutaneous tissues and lungs. Normally cycles in sub-saharan Africa but may be carried into Europe and the Middle East by wind-borne spread of midges. Affects horse, donkeys and mules. Indigenous equidae undergo clinically inapparent or sub-acute infections. Horses imported into endemic areas are likely to die if not vaccinated.

**Clinical features**

Incubation 5-7 days (range 2-21). Four clinical forms:

1. Acute pulmonary syndrome
   Short course of a few hours duration, high fever, conjunctivitis and pulmonary edema with frothy nasal discharge.

2. Sub-acute systemic circulatory form.
   Course is several days. Fever, injected conjunctiva with petechiae, cyanosis of tongue and gums. Initial edematous bulging of supra-orbital fossa, eventually affecting whole head

3. Mixed form with lung edema and sub-cutaneous edema most common.

4. Simple fever seen in horses indigenous to Africa.

**Pathology**

- Gastritis
- Hydrothorax and massive pulmonary edema
- Subcutaneous and subserous edema
- Subepicardial and subendocardial hemorrhages.

**Diagnosis**

- History of travel from endemic area
- Typical clinico-pathological appearance
- Confirmatory AGID using spleen as antigen source.

**Diff. Diagnosis**

- Any cause of sudden death
- Equine babesiosis
- Equine arteritis
- Anthrax

**Vaccine**

- Live attenuated polyvalent vaccine

**Avian Influenza (Fowl Plague)**
There are many avian influenza subtypes and most of them cause no disease or only a mild transient disease. However, sporadic outbreaks of highly pathogenic avian influenza (HPAI) occur worldwide, and have the ability to cause major epidemics if not diagnosed and controlled. Wild birds are a reservoir for the avian influenza viruses. The chief domestic hosts are chickens and turkeys. Outbreaks of HPAI demonstrate high mortality. Clinical signs include sudden onset of illness, dullness, ruffled feathers and a precipitous fall in egg production. There is typically excessive lacrimation and edema of the head and face, especially the comb and wattles, which are cyanotic. There is respiratory distress and diarrhea. Less virulent strains cause depression, drop in egg production, respiratory disease, and sinusitis.

**Differential Diagnosis**
1. Highly virulent viscerotropic Newcastle Disease
2. Fowl cholera
3. Infectious bronchitis
4. Infectious laryngotracheitis
5. Chlamydiosis
6. Chronic respiratory disease (*M. gallisepticum*)
7. Other acute bacterial diseases

**Post mortem findings**
Congestive and hemorrhagic lesions are found in the skin, liver, spleen, heart kidneys and lungs. Low virulence strains cause respiratory disease and sinusitis with mucopurulent exudate. High virulence strains cause severe edema particularly of the head, wattles and comb.

**Samples for diagnosis**
Diagnosis depends on isolation of the virus from tracheal and cloacal swabs of live birds or from the organs of dead birds. The swabs should be inserted deeply to obtain sufficient epithelial material. Samples of blood, sinus exudates, lung, trachea, spleen, intestine, kidneys heart and bone are recommended. All sample should be sent on ice, or frozen if necessary. If it is available, brain heart infusion broth is a good transport medium for tissues and swabs. Blood samples should be submitted for serological diagnosis.

**Newcastle Disease:**
(Exotic Newcastle Disease, Velogenic Viscerotropic Newcastle Disease, VVND)

This is a contagious disease of chickens caused by a paramyxovirus. The disease may manifest itself in different forms affecting the digestive, respiratory and/or nervous systems. The main means of transmission of the virus is by the aerosol route. The incubation period varies from 2 - 15 days and averages 5 – 6 days. Symptoms vary from acute, causing high mortality, to a mild condition which may not be noticed. Clinical signs include diarrhea, respiratory distress, coughing, gasping, nervous signs, paralysis of limbs and torticollis.
Differential Diagnosis:
   1. Infectious Bronchitis
   2. Mycoplasmosis
   3. Marek's Disease
   4. Avian Encephalomyelitis.

Post mortem findings
Gross pathological changes include hemorrhagic/necrotic changes in the intestine and the cecal tonsil, hemorrhages in the proventriculus and trachea and spleen enlargement.

Samples for diagnosis
Laboratory diagnosis relies on the isolation and identification of the causal virus. Live chicks and carcasses should be sent to the laboratory for necropsy. If this is not possible, submit spleens on ice or frozen for virus isolation. Newcastle Disease virus is able to absorb to the surface of red blood cells and induce their agglutination. This property is used to characterize NCD virus. Newcastle Disease specific antiserum inhibits this property. This is the basis of Hemagglutination and Hemagglutination Inhibition Tests for identification of NCD virus.

**African Swine Fever**

This is a viral disease affecting domestic pigs. In Africa, the natural reservoir hosts are wild pigs, especially warthogs and bush pigs, and soft ticks (especially *Ornithodorus moubata*). Acute clinical cases have fever, nasal and ocular discharges, muscle tremors, convulsions, bleeding and dysentery.

Post mortem findings
Widespread hemorrhages are the most striking post mortem feature because the virus severely affects clotting mechanisms. Lymph nodes and spleen are enlarged and hemorrhagic. Hemorrhages may be seen throughout the body. The contents of the gut are often bloody.

Differential Diagnosis:
1. Classical Swine Fever (Hog Cholera)
2. Acute Erysipelas
3. Salmonellosis and other septicemias.

Samples for diagnosis
1. Blood collected into EDTA for virus isolation.
2. Fresh spleen, tonsils and lymph nodes on ice, for virus isolation. If fresh samples cannot be delivered to the laboratory on ice, small specimens may be preserved in 50% glycerol saline and submitted to the laboratory as soon as possible.

**Classical Swine Fever (Hog Cholera)**

Hog cholera (HC) is a highly contagious viral disease of swine that occurs in an acute, a subacute, a chronic, or a persistent form. In the acute form, the disease is characterized by high fever, severe depression, multiple superficial and internal hemorrhages, and high morbidity and mortality. In the chronic form, the signs of depression, anorexia, and fever
are less severe than in the acute form, and recovery is occasionally seen in mature animals. Transplacental infection with viral strains of low virulence often results in persistently infected piglets, which constitute a major cause of virus dissemination to noninfected farms.

Although minor antigenic variants of hog cholera virus (HCV) have been reported, there is only one serotype. Hog cholera virus is a lipid-enveloped pathogen belonging to the family Flaviviridae, genus Pestivirus. The organism has a close antigenic relationship with the bovine viral diarrhea virus (BVDV) and the border disease virus (BDV), as demonstrated in the immunodiffusion and immunofluorescence tests.

**Transmission**

The pig is the only natural reservoir of HCV. Blood, tissues, secretions and excretions from an infected animal contain HCV. Transmission occurs mostly by the oral route, though infection can occur through the conjunctiva, mucous membrane, skin abrasion, insemination, and percutaneous blood transfer (e.g., common needle, contaminated instruments). Airborne transmission is not thought to be important in the epizootiology of HC, but such transmission could occur between mechanically ventilated units within close proximity to each other.

Introduction of infected pigs is the principal source of infection in HC-free herds. Farming activities such as auction sales, livestock shows, visits by feed dealers, and rendering trucks are also potential sources of contagion. Feeding of raw or insufficiently cooked garbage is a potent source of HCV. During the warm season, HCV may be carried mechanically by insect vectors that are common to the farm environment. There is no evidence, however, that HCV replicates in invertebrate vectors. Husbandry methods also play an important role in HC transmission. Large breeding units (100 sows) have a higher risk of recycling infection than small herds. In large breeding units where continuous farrowing is practiced, strains of low virulence may be perpetuated indefinitely until the cycle is interrupted by stamping-out procedures and a thorough cleaning and disinfection are carried out.

**Incubation Period**

The incubation period is usually 3 to 4 days but can range from 2 to 14 days.

**Clinical Signs**

The clinical signs of HC are determined by the virulence of the strain and the susceptibility of the host pigs. Virulent strains cause the acute form of the disease, whereas strains of low virulence induce a relatively high proportion of chronic infections that may be inapparent or atypical. These strains are also responsible for the "carrier-sow" syndrome from which persistently infected piglets are produced.

**Acute Hog Cholera**
In acute HC, the pigs look and act sick. Their disease progresses to death within 10 to 15 days, and remissions are rare. In an affected herd, some pigs will become drowsy and inactive and will stand with arched backs. Other pigs will stand with drooping heads and straight tails. Some pigs may vomit a yellow fluid containing bile. The sick pigs will huddle and pile up on each other in the warmest corner of the enclosure and will rise only if prompted vigorously. Anorexia and constipation will accompany a high fever that may reach 108° F (42.2° C) with an average of 106° F (41.1° C). Pigs may continue to drink and may have diarrhea toward the end of the disease process. Conjunctivitis is frequent and is manifested by encrustation of the eyelids and the presence of dirty streaks below the eyes caused by the accumulation of dust and feed particles. Sick pigs become gaunt and have a weak, staggering gait related to posterior weakness. In terminal stages, pigs will become recumbent, and convulsions may occur shortly before death. In the terminal stage, a purplish discoloration of the skin may be seen; if present, the lesions are most numerous on the abdomen and the inner aspects of the thighs.

**Gross lesions**

The most common lesion observed in pigs dying of acute HC is hemorrhage. Externally, a purplish discoloration of the skin is the first observation. There may be necrotic foci in the tonsils. Internally, the submandibular and pharyngeal lymph nodes are the first to be affected and become swollen owing to edema and hemorrhage. Because of the structure of the pig lymph node, hemorrhages are located at the periphery of the node. As the disease progresses, the hemorrhage and edema will spread to other lymph nodes. The surface of the spleen, and particularly the edge of the organ, may have raised, dark wedge-shaped areas. These are called splenic infarcts. Infarcts are frequently observed in pigs infected experimentally with older strains of HCV but are less commonly seen with the contemporary strains.

Pinpoint to ecchymotic hemorrhages on the surface of the kidney are very common in HC. Such lesions are easier to see in the decapsulated kidney. Hemorrhages are also found on the surface of the small and large intestine, the larynx, the heart, the epiglottis, and the fascia lata of the back muscles. All serous and mucosal surfaces may have petechial or ecchymotic hemorrhages.

**Rinderpest** (*believed eradicated from the face of the earth as of 2010*)

As an aid in Rinderpest surveillance and to make sure that all outbreaks of Rinderpest are investigated, the Global Rinderpest Eradication Program has created a stomatitis-enteritis case definition which includes all the differential diagnoses for Rinderpest. This is not a definition of Rinderpest itself but a definition of outbreaks which must be investigated with the highest priority, in order that no outbreaks of Rinderpest are missed.
### Stomatitis-Enteritis Case Definition

<table>
<thead>
<tr>
<th>Symptom</th>
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<tr>
<td>Ocular Discharge</td>
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<td>Nasal Discharge</td>
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<td>And</td>
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<tr>
<td>Fever</td>
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<tr>
<td>Oral Erosions/Lesions</td>
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<td>Salivation</td>
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<td>Corneal Opacity</td>
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<tr>
<td>Diarrhea</td>
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<tr>
<td>Death</td>
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</tbody>
</table>

Outbreaks of contagious disease exhibiting discharge (ocular and nasal) and any two of the above-mentioned symptoms should be reported as stomatitis-enteritis outbreaks. Note that it is the outbreak that must meet the case definition, not individual animals.

Rinderpest is a virus disease causing fever, stomatitis, gastroenteritis, dehydration and death in ruminants. Transmission is by close contact between healthy and infected animals. Clinical signs include fever, necrotic erosions in the mouth and vagina, nasal and ocular discharges, diarrhea, dehydration and death. Subacute cases are less severe and one or more of the classic clinical signs may be missing. In sheep and goats pneumonic signs are more prominent and the oral erosions may not be obvious.

**Differential Diagnosis:**
2. Peste des Petits Ruminants
3. Foot and Mouth Disease
4. Malignant Catarrhal Fever.
5. Bluetongue

**Post mortem findings:**
Post mortem examination reveals mucosal erosions, hemorrhagic gastroenteritis, necrosis of Peyer's patches and cecal tonsil, and hemorrhages along the mucosal folds of the colon (zebra striping).

**Samples for diagnosis**
Laboratory diagnosis is based on the identification of specific Rinderpest viral antigens in tissues and secretions, and less commonly on virus isolation and antibody titers. All outbreaks which fit the stomatitis/enteritis case definition should be investigated and treated as Rinderpest suspects. It is important to collect samples from a number of animals, not just one animal. Animals in early stages of the disease are much better sample donors than animals that have been sick for a long time or have died.
**Bluetongue**
Bluetongue is an *Orbivirus* caused disease of sheep (and occasionally other ruminants) transmitted by Culicoides midges. There are at least 25 serotypes world-wide. Infection of cattle, goats and camels is usually without clinical signs. Affected animals show fever, nasal discharge, swelling and necrotic ulceration of the tongue, gums and lips, and sometimes in the later stages, lameness due to myositis or laminitis and coronitis. Secondary pneumonia often follows. The mortality rate is variable.

**Differential Diagnosis:**
1. Foot and Mouth Disease
2. Peste des Petits Ruminants
3. Contagious Ecthyma (Orf)
4. Sheep pox
5. Epizootic Hemorrhagic Disease (EHD)

**Post mortem findings:**
Ulceration of nasal and oral mucosa, variable enteritis, and hemorrhages of the skeletal, heart and tongue muscle.

**Epizootic Hemorrhagic Disease of Deer**
This disease is caused by an *Orbivirus* closely related to Bluetongue Virus and is also transmitted by *Culicoides* midges. It cross reacts with BT in the gel diffusion test but can be distinguished by virus neutralization. It occurs sporadically in Indiana. The last outbreak in 1996 affected mainly older beef cows and some calves, and white tail deer. Affected animals are febrile, drooling, and have reddening and necrotic ulceration of the oral mucosa. There may be cracking and ulceration of the skin of the teats. The feet are exquisitely painful and the animals are reluctant to move and often stand in ponds and water courses. The lesions are of an immune mediated vasculitis. Mortality is high in deer (up to 90%) but low in cattle which recover in about 5 days.

**Contagious Bovine Pleuro-Pneumonia**
This is a contagious pneumonic disease of cattle caused by *Mycoplasma mycoides* var *mycoides*. Affected animals suffer persistent coughing, difficulty breathing, and discharges from the nose and mouth. The disease causes pneumonia, serofibrinous pleurisy, fluid in the chest cavities and interlobular edema of the lungs. Acutely affected cattle show fever, rapid respiration rate, anorexia and depression. A cough develops, breathing becomes difficult and nasal and oral discharges are seen. Chronically affected animals show less marked symptoms. Calves under 6 months of age are more likely to have swollen painful joints than pneumonia. Many animals will recover but remain dangerous carriers of the disease.

**Differential Diagnosis:**
1. Hemorrhagic Septicemia
2. Bacterial or viral broncho-pneumonia
3. Theileriosis.
Post mortem findings
The affected lung adheres to the pleura by a yellow fibrinous tissue. The pleural cavity is filled with a turbid yellow exudate. The cut surface of the lung shows a marbled appearance due to distension of the interlobular septae with serofibrinous exudates. In chronic cases an area of dead lung tissue surrounded by a fibrous capsule (sequestrum) may be seen.

Samples for diagnosis
Laboratory confirmation depends on detection of the organism or of serum antibodies.

1. Send portions of affected lung and pleural exudate, collected aseptically from the chest cavity, either fresh or preserved in 50% glycerol saline. Refrigerate all specimens.
2. Blocks of lung tissue, preserved in 10% formol saline should be sent for histopathology and immuno-histochemistry.
3. Infection can be confirmed serologically by means of the complement fixation test or ELISA test. Sera from suspects should be sent frozen.
4. A rapid slide agglutination test which detects antibodies may be available as a field test.

Contagious Caprine Pleuro-Pneumonia
This is a peracute, acute or chronic disease of goats caused by Mycoplasma mycoides var. capri. The main features of the condition are fibrinous pneumonia, pleurisy and pleural exudate. Acute cases have fever and pneumonia. Chronically affected goats show nasal catarrh, cough, enteritis and general debility.

Differential Diagnosis:
1. Pneumonias - bacterial and viral.

Post mortem findings
Pneumonic areas of lung are covered with a yellowish deposit. Yellow fluid is found in the chest cavity. Adhesions between lung and pleura are seen.

Samples for diagnosis
Lung, lymph nodes and pleural exudate should be sent fresh (or in 50% glycerol saline) on ice for mycoplasma isolation or antigen detection. Blocks of affected lung and mediastinal lymph node should be preserved in 10% formalin for histopathology.

Malignant Catarrhal Fever
This is an acute, fatal viral infection of cattle, buffalo and deer, caused by infection with alcelaphine herpesvirus-1 (wildebeest associated) and ovine herpesvirus-2 (sheep associated). The two syndromes are clinically indistinguishable and are characterized by fever, necrosis of oral and nasal mucosa, lymphadenopathy, kerato-conjunctivitis and blepharospasm. Affected animals are severely depressed, have profuse muco-purulent nasal discharge, ocular discharge and often severe dyspnea. Nervous signs often occur. Cattle are infected by contact with sheep (or wildebeest), which are symptomless carriers.
The disease is not contagious among cattle. Outbreaks sometimes occur without a history of contact with sheep.

**Differential Diagnosis:**
1. Rinderpest
2. Bovine Virus Diarrhea/ Mucosal Disease
3. Infectious bovine rhinotracheitis (IBR)

**Post mortem findings**
There is inflammation and necrosis of the mouth and alimentary tract. If diarrhea is present hemorrhages along the mucosal folds of the colon (zebra striping) may be seen. Spleen and lymph nodes are enlarged, the liver is swollen and hemorrhages are seen in other organs. Opacity of the cornea is always present.

**Samples for diagnosis**
Confirmation of Malignant Catarrhal Fever is by histopathological demonstration of a necrotizing vasculitis. Submit intestinal mucosa, lymph node, brain, kidney and other organs in 10% formol-saline, and an eye fixed in Bouin’s solution. Virus isolation is not usually attempted but detection of virus nucleic acid by PCR may be available, and requires submission of lymph node, spleen and lung on ice.

**Lumpy Skin Disease**
Lumpy Skin Disease is an acute arthropod-borne viral disease of cattle. The causative virus (Neethling virus) is related to the pox virus of sheep and goats. During a 1980/81 outbreak in Sudan morbidity rates of up to 60% and mortality rates of up to 10% were reported. Affected animals show fever, depression, conjunctivitis, intradermal cutaneous nodules, swelling of lymph nodes and edema of the dependent parts.

**Differential Diagnosis:**
1. Malignant Catarrhal Fever.
2. Pseudo Lumpy Skin Disease (Allerton virus).
3. Photosensitization.
4. Cutaneous pustular lesions of tropical theileriosis
5. Cutaneous streptothricosis

**Post mortem findings:**
The intradermal nodules (5-70 mm in diameter) which form all over the body, are firm and circumscribed. The nodules may become necrotic and form “sitfasts,” which heal only slowly and leave a scar. Nodules are found in the viscera of animals that die, particularly in the respiratory tract and lungs. Lymph nodes are enlarged.

**Samples for diagnosis**
Live animals: Take a biopsy sample from an early cutaneous nodule, place half the specimen in 10% formalin and freeze the other half. Send both samples to the laboratory. The formalized tissue will be examined histologically for the presence of intracytoplasmic inclusion bodies. The frozen nodule will be processed for virus isolation or electron microscopy.

Dead animals: Send cutaneous and visceral nodes, and pieces of lymph node in formalin, as well as frozen samples.